

DESCRIPTION

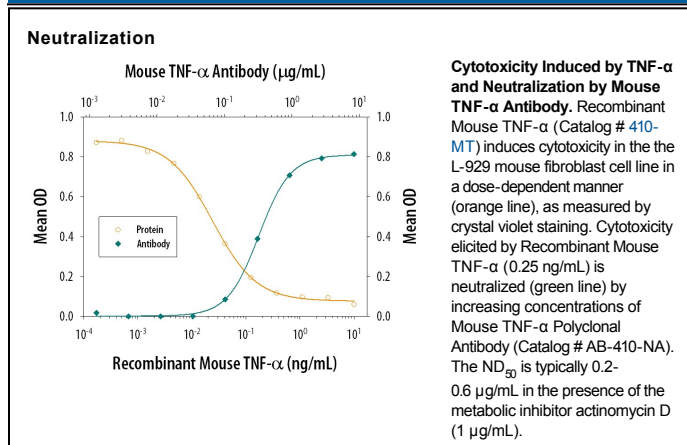
Species Reactivity	Mouse
Specificity	Detects mouse TNF- α in direct ELISAs and Western blots. In direct ELISAs, approximately 15% cross-reactivity with human TNF- α is observed, and less than 5% cross-reactivity with recombinant bovine TNF- α , recombinant canine TNF- α , recombinant equine TNF- α , recombinant feline TNF- α , and recombinant porcine TNF- α is observed.
Source	Polyclonal Goat IgG
Purification	Protein A or G purified
Immunogen	<i>E. coli</i> -derived recombinant mouse TNF- α Leu80-Leu235 Accession # P06804
Endotoxin Level	<0.10 EU per 1 μ g of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 μ m filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	1 μ g/mL	Recombinant Mouse TNF- α (Catalog # 410-MT)
Neutralization	Measured by its ability to neutralize TNF- α -induced cytotoxicity in the L-929 mouse fibroblast cell line [Matthews, N. and M.L. Neale (1987) in <i>Lymphokines and Interferons, A Practical Approach</i> . Clemens, M.J. <i>et al.</i> (eds): IRL Press. 221]. The Neutralization Dose (ND ₅₀) is typically 0.2-0.6 μ g/mL in the presence of 0.25 ng/mL Recombinant Mouse TNF- α and 1 μ g/mL actinomycin D.	

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 1 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <ul style="list-style-type: none"> • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Tumor necrosis factor alpha (TNF- α , also known as cachectin and TNFSF2, is the prototypic ligand of the TNF superfamily. It is a pleiotropic molecule that plays a central role in inflammation, apoptosis, and immune system development. TNF- α is produced by a wide variety of immune and epithelial cell types (1, 2). Mouse TNF- α consists of a 35 amino acid (aa) cytoplasmic domain, a 21 aa transmembrane segment, and a 179 aa extracellular domain (ECD) (3). Within the ECD, mouse TNF- α shares 94% aa sequence identity with rat and 70% - 77% with bovine, canine, cotton rat, equine, feline, human, porcine, rat, and rhesus TNF- α . The 26 kDa type 2 transmembrane protein is assembled intracellularly to form a noncovalently linked homotrimer (4). Ligation of this complex induces reverse signaling that promotes lymphocyte costimulation but diminishes monocyte responsiveness (5). Cleavage of membrane bound TNF- α by TACE/ADAM17 releases a 55 kDa soluble trimeric form of TNF- α (6, 7). TNF- α trimers bind the ubiquitous TNF RI and the hematopoietic cell-restricted TNF RII, both of which are also expressed as homotrimers (1, 8). TNF- α regulates lymphoid tissue development through control of apoptosis (2). It also promotes inflammatory responses by inducing the activation of vascular endothelial cells and macrophages (2). TNF- α is a key cytokine in the development of several inflammatory disorders (9). It contributes to the development of type 2 diabetes through its effects on insulin resistance and fatty acid metabolism (10, 11).

References:

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